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Computational investigation of nsSNPs in Monoamine Oxidase B (MAOB) protein

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Abstract

Monoamine Oxidase B (MAOB) mutation is one of the genetic causes for the Parkinson's disease. Mutation in this gene leads to increased degradation of phenylthylamine, benzylamine and dopamine. The reduce in neurotransmission of dopamine leads to the cognitive decline. Non-synonymous SNPs (nsSNPs) are typical genetic variants that may potentially regulate the expression of proteins in high level. The aim of the present study was to evaluate the phenotypic effect of nsSNPs in polymorphic variants of MAOB genes. The number of possible nonsynonymous SNPs (nsSNP) present in MAOB was analyzed using Galaxy online data analysis platform and the sequence variant effects in the gene of interest were performed by using SIFT and PolyPhen2 tools. It was found that about 39.3 % of the nsSNPs were deleterious. Some of the mutations which involve in the Parkinson's disease includes c1.437666834,1,G/T (V35G), c1.437667553,1,T/G (V66G) and c1.438666050,1,G/T (D164Y) found to exert benign effects on the protein structure and function and were chosen for further analysis. Protein structural analysis with these amino acid variants was performed by I-mutant, Project Hope and Rasmol. Our In-silico analysis suggested that D164Y and V35G variants of MAOB could lead to functional deviations of protein to some extent.

Keywords: MAOB, nsSNP, Parkinson's disease, variant analysis

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